In the Specification:

On page 1, before the "Background of the Invention," please insert the following paragraph:

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--**Related Applications**

This application claims priority to U.S. Provisional Application No.: 60/192,727, filed on March 27, 2001, incorporated by reference herein in its entirety.--

Replace the paragraph at page 4, lines 8-27, with the following paragraph:

~~--*Figure 1 shows*~~ *Figures 1A-D* show FcαRI expression on Kupffer cells. (a) Paraffin liver sections of G-CSF treated NTg mice (left panel), and untreated (middle panel), or G-CSF treated (right panel) CD89 Tg mice were stained for expression of human FcαRI. Bar represents 30 μm (pictures taken with objective 40x; *inset right* panel, objective 100x). Only in G-CSF treated Tg mice cytoplasmic staining for FcαRI was found in stellate-shaped cells, lining the liver sinusoids. This experiment was repeated five times with similar results. (b) A double staining for both FcαRI

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and a macrophage marker (F4/80) was performed to identify stellate cells as Kupffer cells. F4/80 and FcαRI immunoreactivity are shown in blue and red, respectively, as described in the methods section. Kupffer cells of G-CSF treated Tg mice stain positive for FcαRI (right panel; cells are both blue and red), whereas Kupffer cells in NTg mouse livers are negative for FcαRI (left panel; only blue staining). Bar represents 50 μm (objective 20x). (c) Isolated Kupffer cells of G-CSF treated Tg (red line), and NTg control littermates (black line) were stained with Pe-labeled anti-FcaRI mAb A59 (Monteiro, R.C., *et al.* J. *Immunol. 148,* 176-1770 (1992)) and analyzed by flow cytometry, showing positive staining of Tg Kupffer cells. (d) Expression of FcαRI on human Kupffer cells. A liver sample of a patient with active viral Hepatitis type C is shown (cryo section). Sections were stained for both CD68, a human macrophage marker (blue), and FcαRI (red) Left panel: negative control (anti-FcαRI Ab omitted). The right panel shows positive Kupffer cells. Bar represents 30 μm (objective 40x; inset, objective 100x).--

Replace the paragraph at page 4, line 29 through page 5, line 5, with the following paragraph:

-- ~~*Figure 2* shows~~ *Figures 2A-D* show that Kupffer cells expressing FcαRI mediate phagocytosis of IgA-coated bacteria FITC labeled. Serum IgA opsonized *E. coli* bacteria were injected i.v. into G-CSF-treated mice. Mice were sacrificed and liver section taken. (a) Fluorescence of NTg (left panel) and Tg (right panel) liver sections was analyzed with fluorescence microscopy. Bar represents 50 μm (objective 20x; inset

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right panel objective 40x). (b) FITC fluorescence of Tg liver sections was determined (left panel), before staining with F4/80 mAb (middle panel; red) to identify fluorescent stellate cells as Kupffer cells. A computerized overlay picture of both images (right panel) was produced demonstrating fluorescent IgA-coated bacteria to co-localize with F4/80 positive Kupffer cells. Bar represents 50 μm (c) Confocal microscopy pictures showing two different cell layers confirmed that bacteria are phagocytosed by Tg Kupffer cells. Bar represents 15 μm. (d) Numbers of FITC-fluorescent Kupffer cells of NTg (open bar) and Tg (dotted bar) mice were quantitated by microscopy. Data (mean ± SD) are representative of results obtained in three separate experiments. * $p<0.01$.--

Replace the paragraph at page 5, lines 7-32, with the following paragraph:

-- ~~*Figure 3* shows~~ *Figures 3A-H* show that serum IgA mediates phagocytosis via FcαRI in contrast to secretory IgA (SIgA). (a) Non-reduced SDS-PAGE analysis of IgA preparations; lane 1: serum IgA (ICN,), lane 2: SIgA (ICN,), lane 3: SIgA (Sigma). Molecular weight markers are indicated on the left (m) Proteins were stained with Coomassie Brilliant Blue. (b) E *coli* bacteria

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were incubated with PBS or IgA preparations, washed and stained with PE-labeled $F(ab')_2$ fragments of goat anti-human IgG (left panel) or IgA (right panel) antiserum. Fluorescence was analyzed by flow cytometry, showing all preparations to certain similar amounts of IgA Ab directed against *E. coli*. Black line; PBS, red line; serum IgA, blue line; SIgA ICN, yellow line; SIgA Sigma (c) NTg or Tg PMN were incubated with FITC-labeled, IgA-opsonized *E. coli* bacteria. FITC fluorescence of PMN, reflecting bacterial uptake, was analyzed by flow cytometry. Black line; NTg + serum IgA, yellow line; NTg + SIgA ICN, pink line; NTg + SIgA Sigma. red line; Tg + serum IgA, brown line; Tg + SIgA ICN, blue line; Tg + SIgA Sigma. In addition, Tg PMN were incubated with CD89-blocking mAb My432[21] prior to incubation with serum IgA- coated bacteria; green line. A representative experiment out of-four is shown. (d-f) Human PMN were incubated with IgA

opsonized *E. coli* bacteria (see c), and analyzed by flow cytometry (d, open bars; without blocking mAb, dotted bars; with blocking mAb My43). Cytospin preparations were analyzed with light (e) or confocal microscopy (f; PMN incubated with serum IgA-coated bacteria: three cell layers). Bar represents 10 μm. (g) G-CSF treated Tg mice were injected i.v. with FITC-labeled, SIgA- (left panel) or serum IgA- (right panel)

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opsonized *E. coli* bacteria. Fluorescence of liver sections was analyzed with fluorescence microscopy. Bar represents 50 μm (objective 20x). (h) Respiratory burst activity of human PMN stimulated with serum IgA (red line), SIgA ICN (green line), SIgA Sigma (blue line) or PBS (brown line). This experiment was repeated three times yielding similar results.--